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(10) Mark E. Speckhard, Lieutenant Colonel, USAF, MC

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Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas 78235

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Mark E. Speckhard

MARK E. SPECKHARD, Lt Col, USAF, MC
Project Scientist

Paul T. Hansen

PAUL T. HANSEN, Lt Col, USAF, MC
Supervisor

Robert G. McIver

ROBERT G. MCIVER
Brigadier General, USAF, MC
Commander

UNCLASSIFIED

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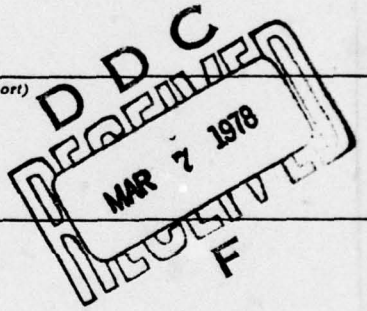

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Altitude decompression sickness (DCS) was once a major cause of incapacitation in aviators. Preventive measures, including cabin pressurization and denitrogenation, have markedly reduced the incidence of altitude DCS. Most flight surgeons will now see only an infrequent case. Despite limited first-hand experience, the physician who cares for aviators must maintain expertise in the diagnosis and management of altitude DCS. The prognosis associated with expeditious primary care and prompt hyperbaric therapy is excellent. This paper reviews with the flight surgeon concepts in the primary care of altitude DCS.

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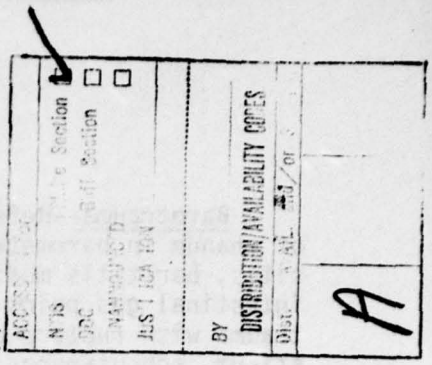

ALTITUDE DECOMPRESSION SICKNESS:

REVIEW OF CONCEPTS IN PRIMARY CARE

INTRODUCTION

A thorough knowledge of the manifestations and management of altitude decompression sickness (DCS) is essential for the physician who cares for aviators. Prior to 1959 many thousands of aviators were incapacitated and occasionally killed by altitude DCS. In 1959 a new era in management was initiated by Donnell and Norton (12) when they successfully treated a case of altitude DCS with compression therapy. Since 1959 we know of no fatalities and only one case with a residual deficit from altitude DCS in the United States Air Force (10).

Excellent historical reviews document the gradual evolution in our understanding of the pathophysiology of altitude DCS (1, 4, 26). We have learned to recognize its often atypical manifestations, and we have come to appreciate the excellent prognosis associated with prompt compression therapy. Cabin pressurization and denitrogenation have proved to be highly successful preventive measures. With increased understanding of altitude DCS and effective prevention the incidence of altitude DCS has dropped precipitously.

Success in the management of altitude DCS has produced a new challenge. The flight surgeon who rarely sees a case of altitude DCS must remain keenly aware of its presence, must be alert to its manifestations, and must be knowledgeable in its management. This paper reviews with the flight surgeon current concepts in the primary care of altitude DCS. Additional background and perspective can be gained from general discussions of altitude DCS currently in the literature (7, 16, 17, 25, 31), as well as from the individual references cited.

TERMINOLOGY

Decompression Sickness (DCS)--Refers to disease due to evolved gas in the tissues resulting from a reduction in barometric pressure. In the past, and to some extent today, the term "decompression sickness" was used interchangeably with "the bends." In our discussion we have joined common usage by limiting the definition of bends to that manifestation of DCS which involves only pain and occurs in the musculoskeletal system. To emphasize that we are using bends in this limited fashion we refer to it as pain only bends.

Barotrauma--Refers to disease or symptoms resulting from the effect of change in barometric pressure on gas trapped in the body. Barosinusitis, barotitis media, pulmonary barotrauma, aerodontalgia, and gastrointestinal gas pains are manifestations of barotrauma. Pulmonary barotrauma with rupture of alveoli may result in pneumothorax, pneumomediastinum, subcutaneous emphysema, or air embolism.

Air Embolism--Refers to disease resulting from the introduction of a bolus of air into the circulatory system. Air embolism may result from pulmonary barotrauma or may result from the accidental introduction of air into the circulatory system at surgery or during venoclysis. The risk of pulmonary barotrauma with secondary air embolism is exceedingly small in the aerospace environment in contrast to its much more frequent occurrence undersea. Air embolism frequently affects cerebral circulation where it usually results in immediate, dramatic, and profound neurologic deficit which is often associated with sudden loss of consciousness. The etiology and pathophysiology of air embolism are distinct from DCS, and the manifestations are rarely confused.

PATHOPHYSIOLOGY

The etiology of DCS is incompletely understood. Bubbles evolved by the tissues under the influence of decreased barometric pressure play a central role. The blood is probably the major tissue involved in the evolution of bubbles, but gas can be evolved by other tissues and has been demonstrated in tendon sheaths and joint cavities.

Two factors essential for bubble formation are supersaturation of tissues by gas and the presence of gas nuclei. Tissues become saturated with gas in direct proportion to the environmental pressure. This is in accordance with Henry's Law which states that the concentration of gas dissolved in a liquid is directly proportional to the pressure of the gas to which the liquid is exposed. With a decrease in environmental pressure a lesser amount of gas is sufficient to saturate the tissues. If the environmental pressure decrease is gradual, excess gas in the tissues can diffuse into the blood where it can further diffuse into pulmonary alveoli and be exhaled. If the environmental pressure change is large enough and rapid enough to exceed the capabilities of this physiologic mechanism, tissues which were saturated will become supersaturated. Supersaturation will be greatest in tissues with the least blood supply and with the greatest dissolved nitrogen content, such as fat.

Gas nuclei are minute collections of gas which differ from bubbles only in size. The boundary between gas nuclei and bubbles is arbitrary. While the origin of gas nuclei continues to be studied, we know that gas nuclei arise more easily in turbulent blood flow, in damaged tissues, in tendons and joints, and in contracting muscles.

The tendency for gas nuclei to develop in selected areas is manifest clinically in several ways. Although evidence is anecdotal, people with recent injuries such as fractures, sprains, or contusions may be more susceptible to DCS; and persons with old injuries may have a predilection for DCS to localize at the sites of old injury. The shearing action of tendons, ligaments, and joints is a likely mechanism for gas nuclei production and may account for the increased incidence of DCS around joints. Production of gas nuclei in contracting muscles is thought to be related to increased carbon dioxide tension. This production of gas nuclei may explain why exercise predisposes to DCS.

Bubbles are composed of a mixture of nitrogen, oxygen, carbon dioxide and water vapor. Nitrogen is the major component and most troublesome since it is inert and not readily resorbed. Bubbles forming in the blood arise in the capillary and venous circulation since the increased pressure in the arterial system is less conducive to bubble formation (7). Bubbles in the capillaries and veins block venous outflow from tissue, resulting in stagnation, hypoxia, and subsequent edema. Within hours of bubble formation a coat of fibrin may develop around each bubble. Prompt treatment of DCS is aimed at resolution of bubbles prior to fibrin formation and before increasing hypoxia and subsequent edema make resolution more difficult and more prolonged.

There is much discussion in the literature over the role of mechanisms other than the mechanical effect of bubbles in the etiology of DCS. Several factors support the concept that multiple etiologic mechanisms are involved: First, there is considerable variability in the susceptibility to DCS among individuals and in the same individual on different occasions, which is not well explained if one relies solely on the mechanical effects of bubbles in the etiology of DCS. Second, some cases of DCS have been resistant to compression therapy. This resistance is more easily explained if there are multiple etiologic factors in addition to the mechanical effect of bubbles. Third, multiple changes in the blood and vascular system have been demonstrated in DCS. Investigators have reported on and discussed the role of sludging of red blood cells (13, 19), platelet agglutination and thrombocytopenia (18, 20, 21, 23), elevated serum enzymes (14, 24, 30), decrease in plasma lipids (28), formation of lipid emboli (8), and fluid and electrolyte shifts (6, 19). Coagulopathy has been observed suggestive of disseminated intravascular coagulation (29). Demonstration of endothelial damage (34) and angiospasm (23) have raised the question of vascular mechanisms in the etiology of DCS.

A critical question is whether blood and vascular abnormalities noted in the laboratory play a role in the causation of DCS or are simply a reflection of disease caused by the mechanical effect of bubbles. At issue is whether prevention or treatment of these alterations will prevent or resolve the signs and symptoms of DCS.

The most telling point in favor of a single mechanical etiology for DCS is the observation that there is complete resolution of all manifestations in most cases of DCS following compression therapy alone. Since compression therapy can account for the resolution of the mechanical effects of bubbles, this observation is most easily explained by a single mechanical etiology of DCS and poses a major problem in establishing the significance of blood-bubble interactions in the pathophysiology of DCS.

This area continues under extensive study to clarify the roles of the mechanical effects of bubbles and blood-bubble interactions in the etiology and pathophysiology of DCS.

RISK FACTORS

Experience indicates a number of risk factors for DCS:

- Altitude
- Diving prior to exposure to altitude
- Duration at altitude
- Age
- Obesity
- Exercise at altitude
- Cold at altitude

Suggested mechanisms by which some of these risk factors predispose to DCS have already been discussed in the section "Pathophysiology."

The altitude to which the individual is exposed is a major risk factor for DCS. In pressurized craft it is of course the cabin altitude rather than the flight altitude which represents the environmental pressure to which the aviator is exposed. The degree of pressure change required to produce DCS was evaluated by the English physiologist Haldane. He noted that halving of the environmental pressure was necessary before bubble formation and DCS would appear. On the basis of this observation one would anticipate that an aviator in equilibrium with air at sea level would be safe from DCS at altitudes up to 18,000 ft (5488 m) (one-half atmospheric pressure), but would be at risk for DCS at altitudes above 18,000 ft (5488 m).

Clinical experience supports this observation. A case of severe DCS occurred at 18,500 ft (5640 m) (15), the lowest altitude reported at which DCS occurred in an individual who had not been exposed previously to greater than sea-level atmospheric pressure. That serious neurologic DCS can occur at minimal altitudes was reemphasized by Davis et al. (11) in the report of cases occurring at 19,000 ft (5793 m) and 28,000 ft (8536 m). Above 18,000 ft (5488 m), increasing altitude is associated with increasing risk for DCS (10).

DCS can occur at less than 18,000 ft (5488 m) in the individual who has been subject to environmental pressures greater than sea-level atmospheric pressure, as occurs in diving or in compression chambers. DCS has occurred in flight at altitudes as low as 5,000 ft (1524 m) under this circumstance. This occurrence has prompted the precaution not to fly for 24 hr following diving or compression chamber exposure.

Duration of exposure to altitude is also a well-recognized major risk factor and the longer the time spent at altitude the greater the risk of DCS.

The rate at which the aviator ascends to altitude is governed by the time it takes an aircraft to climb to altitude, and even with high performance aircraft the rate of ascent does not appreciably affect the risk for DCS. Whether it takes several minutes or several times that long to ascend to altitude, the risk for DCS remains essentially the same. This is in contrast to the undersea environment where multiples of atmospheric pressure can be quickly traversed and the rate of change in environmental pressure is an important risk factor for DCS.

Age and obesity are widely recognized risk factors for altitude DCS, but the degree of risk is not clearly established (7).

Exercise at altitude is a high-risk factor for DCS. Exercise has been a distinguishing feature on many occasions in which one member of a crew has experienced DCS.

Cold is a minor risk factor for DCS but may play a contributory role in an aviator already compromised by other risk factors.

Prior injury is considered a risk factor for DCS by some, but evidence is anecdotal and injury as a risk factor has not been documented by a formal study.

PREVENTION

The two most important factors in the prevention of altitude DCS are denitrogenation and cabin pressurization. Denitrogenation refers to a decrease in the amount of nitrogen absorbed in body tissues, and is usually accomplished by breathing 100% oxygen for a period prior to flight. The aviator then continues to breathe 100% oxygen during flight to prevent reintroduction of nitrogen and to supply oxygen needs.

The need for denitrogenation and time spent at denitrogenation vary with mission requirements. Denitrogenation is usually not necessary when the planned cabin altitude will not exceed 18,000 ft (5488 m). Guidance in the assessment of need for denitrogenation can be obtained through consultation with a hyperbaric physician.

If denitrogenation is interrupted by air breathing, much of its protective value is lost (9). When an aviator develops altitude DCS following denitrogenation, it is useful to determine if denitrogenation was abbreviated or interrupted by air breathing.

Cabin pressurization is the most responsible preventive factor in reducing the incidence of altitude DCS. However, the concept of cabin pressurization, sufficient to prevent DCS, is not applicable to all aircraft or all flight profiles. Some aircraft are not suited to carry the equipment necessary for cabin pressurization. These aircraft are generally limited to flying below 25,000 ft (7620 m) where the risk for DCS is slight but present. Some aircraft, designed to fly at very high altitudes, maintain a degree of cabin pressurization but not sufficient to keep cabin altitudes below 18,000 ft (5488 m). Even in pressurized aircraft accidental loss of pressurization can occur, exposing the occupants to the risk of DCS.

In addition to denitrogenation and cabin pressurization, preventive measures include a reduction of risk factors whenever practicable. Not all risk factors are amenable to intervention. Tests for individual susceptibility to DCS have not provided consistent results (17) and cannot be relied on as an aid in crew selection as a means of avoiding DCS. One can encourage the use of protective clothing to keep exposure to cold at altitude to a minimum. The crewmember who is aware that exercise at altitude is a risk factor for DCS may be able to reduce this factor to a degree that is still consistent with the full performance of his duties. A prime example of risk factor intervention is avoiding flying for 24 hr after diving.

Continued education and physiological training should be utilized to emphasize to flight crews the principles of prevention and initial management of altitude DCS.

MANIFESTATIONS OF ALTITUDE DCS

Manifestations of altitude DCS can be classified as minor (Type I) or major (Type II) as outlined in Table 1.

Cutaneous Manifestations

Pruritus, tingling, or formications may occur without any visible lesions and may be related to gas bubbles in the skin or subcutaneous tissues. Occasionally an erythematous rash may occur with or without these symptoms. There is no clinical significance to these symptoms; they are transitory and do not require treatment. The risk of impending Type II DCS is no greater in the presence of these cutaneous manifestations than in the asymptomatic aviator, so no period of observation is indicated.

TABLE 1. CLASSIFICATION OF MANIFESTATIONS OF ALTITUDE DECOMPRESSION SICKNESS

Minor (Type I)	Major (Type II)
Cutaneous manifestations	Pulmonary manifestations (chokes)
Pain only bends	Neurologic manifestations (staggers)
	Vasomotor collapse

Mottling or marbling of the skin, with or without cyanosis, may occur in the presence of other manifestations of Type II DCS. It carries a serious prognosis and may be a sign of impending vasomotor collapse.

Pain Only Bends

Pain only bends is the most frequent manifestation of altitude DCS. Pain only bends occurs as the sole manifestation of altitude DCS in 60% of cases requiring compression therapy (10). In addition, pain only bends are present in almost half of the remaining 40% of cases. Pain only bends are significant because they may herald impending Type II DCS. Because of this association they require prompt treatment with compression therapy.

Bends pain is usually a dull ache in a joint or deep in the soft tissue or bone; multiple sites may be involved. Knee and shoulder joints are the most frequently affected, although any joint is a potential site. Soft tissue or bone pain most commonly occurs in the extremities. The pain is often vague and may migrate from spot to spot suggesting an hysterical reaction to the uninitiated. The involved area is often tender and may occasionally have slight overlying edema.

The onset of symptoms in pain only bends may occur at altitude or may be delayed. In approximately 90% of cases pain begins within 12 hr of the exposure to altitude. Delayed onset rarely exceeds 24 hr, and initial onset of pain over 36 hr following exposure to altitude should be ascribed to a cause other than DCS.

Symptoms in pain only bends may resolve during descent or spontaneously on the ground. Following initial resolution, pain may recur in a small proportion of cases. The interval is usually short, perhaps a few hours, and in any event would not likely exceed 24 hr.

Pulmonary Manifestations

Pulmonary manifestations, commonly called chokes, are due to multiple pulmonary gas emboli. As in patients with pulmonary emboli from other causes, the situation is often critical and prompt treatment is essential. Chokes occurring without other manifestations of DCS is a rare event. In combination with other manifestations, chokes occurs in 8% of altitude DCS requiring compression therapy (10).

Chest pain, dry cough, and dyspnea form a classic triad of symptoms associated with chokes. Without all three present it is difficult to make the diagnosis. Cyanosis may be present but it is variable. The onset of chokes may be delayed but not longer than 12 hr; and when chokes occur, it is usually within the first 2 hr.

Neurologic Manifestations

Neurologic involvement is a prominent manifestation of DCS, is present in 34% of cases of altitude DCS requiring compression therapy (10), and appears as the sole manifestation in 14% of cases of altitude DCS requiring compression.

In altitude DCS over 90% of the neurologic manifestations are cerebral. This finding is in contrast to the diving experience in which over 90% of neurologic manifestations involve the spinal cord. The most common neurologic manifestations of altitude DCS are visual symptoms and headache. The visual symptoms include scotomata, visual blurring, and blind spots and are similar to the prodromata of migraine. In fact the mechanism may be a bubble-induced migraine syndrome. The headache is commonly occipital.

In addition to these common manifestations almost any variety of neurologic involvement can occur. These manifestations range from spotty motor or sensory deficits and unilateral paresthesias to monoplegias and paraplegias. Findings often do not fit a classic neurologic pattern and may suggest hysteria to the examiner who is unaware that neurologic DCS can present atypically.

Confusion or a change in affect may be the sole manifestation of neurologic DCS. Consequently one must be alert to very subtle neurologic changes. Convulsive disorders may occur early in the course of altitude DCS as a manifestation of severe cerebral involvement. Seizures can also appear later in the course at 24 to 48 hr when they probably result from cerebral edema.

The onset of first neurologic manifestations may be delayed but not longer than 36 hr and most occur within the first few hours. About 50% of neurologic cases start as pain only bends.

Vasomotor Collapse

Vasomotor collapse with profound shock may occur in DCS and is usually irreversible with a poor response to fluid therapy. Collapse probably results from combined neurologic and circulatory factors, and it is usually responsible for the fatalities in DCS.

Shock as the sole manifestation of altitude DCS is a rare event. In combination with other manifestations of DCS, shock occurs in 6% of cases of altitude DCS which require compression therapy (10).

Aseptic Bone Necrosis

Aseptic bone necrosis is an extremely rare late manifestation of altitude DCS in contrast to its more common occurrence in the diving environment.

Laboratory and X-ray Manifestations

In a case of suspected DCS an x-ray search for evidence of bubbles is not very helpful. Serious cases of DCS may show no evidence of bubbles on x-ray. And when bubbles or pockets of air are demonstrated on x-ray, there is no good correlation between their presence and the severity of disease. The Doppler method of bubble detection has been very effective in demonstrating gas bubbles in the circulation, but it remains by and large a research tool.

Despite the multiple alterations in the blood which have been demonstrated in DCS, the changes are too nonspecific to be of value in establishing a diagnosis of DCS.

A shift of fluid from intravascular to extravascular compartments may occur in DCS and warrants following the severely involved patient with frequent hematocrits for evidence of hemoconcentration and impending shock.

DIFFERENTIAL DIAGNOSIS

The diagnosis of altitude DCS is based solely on history and physical examination in most cases. Consultation with a hyperbaric physician may be helpful in doubtful cases.

The differential between pain only bends and minor trauma can be troublesome. When pain occurs at altitude or following a flight, there may be great temptation to attribute it to previously known trauma. The pain in DCS is commonly made worse by exercise or passive movement of

a joint. Pain relieved by exercise is more compatible with a mild sprain or bruise. A helpful test is the application of pressure over the involved area with a blood pressure cuff. Commonly this will temporarily relieve the pain of DCS and will generally not benefit a sprain or bruise.

Whenever the differential between pain only bends and trauma cannot be established to the examiner's satisfaction, it is always better to treat the patient for DCS. Compression therapy will not adversely affect a sprain or bruise and can be decisively advantageous to the patient with DCS. If the diagnosis of pain only bends is dubious, the physician at the hyperbaric facility has the additional option of conducting a test of pressure in the chamber.

Pain only bends may have an associated localized paresthesia. If the paresthesia clears as the pain clears, it should be considered a manifestation of pain only bends and should not be considered to be evidence for neurologic DCS. Paresthesia occurring at altitude or following exposure to altitude, with no pain associated, should be considered a manifestation of neurologic DCS unless another etiology is obvious.

Crossed legs and resting elbows with resultant pressure on nerves can produce paresthesias that are difficult to differentiate from neurologic DCS. Altitude chamber operators routinely precaution against such positions. Unfortunately once paresthesias occur there is no way of resolving this differential unless there is a clear history of nerve pressure. Paresthesias of uncertain etiology should be treated as DCS.

Rarely one may be faced with the differential diagnosis between air embolism and neurologic DCS. In air embolism the onset of neurologic symptoms is usually sudden and dramatic and often profound, while in DCS the onset of neurologic symptoms is usually gradual.

When an aviator presents with pulmonary symptoms, which originated during exposure to altitude, one must distinguish pulmonary manifestations of DCS from "false chokes," which is a dry cough associated with breathing oxygen. The "chokes" of pulmonary DCS are manifest by chest pain, dyspnea, and dry cough. The absence of chest pain and dyspnea in "false chokes" helps distinguish "false chokes" from the pulmonary manifestations of DCS.

PRIMARY CARE OF ALTITUDE DCS

The aim of therapy in DCS is to halt progression of the disease, accomplish prompt resolution of signs and symptoms, and avoid residuals. Hyperbaric therapy is the only definitive treatment of DCS and is effective through the physical effects of increased barometric pressure

and the physiologic effects of associated hyperoxygenation. The increased environmental pressure in the chamber reduces bubble size as a reflection of Boyle's Law, which states that at constant temperature the volume of gas is inversely proportional to the pressure. By reducing bubble size some bubbles will dislodge and allow blood flow to resume. The increased surface tension associated with reduced bubble size may cause some bubbles to collapse and resolve completely. A second physical effect of increased barometric pressure is to aid in tissue absorption of gas from bubbles in accordance with Henry's Law.

During compression therapy for DCS the patient intermittently breathes 100% oxygen at increased barometric pressure. Breathing 100% oxygen provides an increased gradient for washing nitrogen from evolved bubbles and aids in their resorption. The increased gradient also helps wash nitrogen from supersaturated tissues and thus helps prevent further bubble formation. In addition, hyperbaric oxygenation results in an increased partial pressure of oxygen at the tissue level which can hyperoxygenate hypoxic tissue. Hyperoxygenation is also effective in reducing cerebral edema.

A major role of the primary physician is that of determining which patients are candidates for compression therapy. Consultation with a hyperbaric physician can be very helpful in making this decision. The Hyperbaric Center at Brooks Air Force Base, Texas, provides consultation service for both military and civilian physicians. Consultation is provided 24 hr a day and can be obtained by calling commercial telephone 512-536-3278 or AUTOVON 240-3278. Other Air Force hyperbaric facilities are geographically located to provide coverage throughout the United States and in selected overseas locations (3). Since consultation may play an important role in deciding whether a patient requires therapy and is essential in arranging therapy for those who do, the phone number of a hyperbaric consultant deserves a prominent spot in any office where aviators receive care.

The primary care of patients with DCS begins promptly when signs or symptoms first appear. The patient himself, fellow crewmembers, or chamber operators may need to provide initial care. A physician should provide input as soon as possible to evaluate and support the patient, outline a course of therapy, and arrange prompt and safe transportation to a compression chamber when indicated.

Initial Care by the Flight Crew

As soon as symptoms suggestive of DCS occur, the patient or crew should immediately apply 100% oxygen by mask and begin prompt descent. A tight-fitting mask is necessary to provide high concentrations of oxygen. Initially, oxygen should be applied by whatever means are available but a snug-fitting mask should be used as soon as possible.

Increased barometric pressure associated with descent provides an environment less conducive to further bubble formation and may have a beneficial effect on bubbles already formed. Crews should be cautioned not to continue at altitude while attempting to establish the diagnosis with certainty. The mere suspicion of DCS warrants prompt descent unless the conditions of flight make descent impossible or unless mission completion is an overriding factor. Flight crews should be aware that the earliest suggestion of DCS signals the potential for serious disease and possible incapacitation of a crewmember.

Ground-level environment and prompt medical attention are immediate goals; the aircraft should land at the nearest airstrip with a medical facility, whether civilian or military. Once on the ground the patient should receive prompt medical attention, should be kept at strict rest, and should continue to receive 100% oxygen. Symptoms of DCS often resolve during descent, but the crew should not relinquish efforts to reach the nearest medical facility. Symptoms can recur, even at low altitudes or at ground level, and may be severe when they do.

If symptoms are minor or resolve completely, a physician unaccustomed to treating aviators may not realize the potential for exacerbation or recurrence. Consequently even after the patient has received medical care, he should not resume flight until he has been cleared by a flight surgeon or flight medical examiner.

Initial Care by Altitude Chamber Operators

Symptoms suggestive of DCS warrant immediate application of 100% oxygen by snug-fitting aviator's mask and prompt descent of the affected individual to ground level. The patient should be kept at rest and receive prompt medical attention no matter how minor the symptoms.

Primary Care by the Physician

A physician who receives a call from the flight line or the altitude chamber notifying him of a possible case of DCS can initiate primary care over the telephone by advising 100% oxygen and rest. As promptly thereafter as possible the physician should evaluate the patient.

Since prompt therapy is essential for DCS, time spent performing a lengthy history and physical is usually not warranted. On the other hand the physician and his consultant need a data base for determining an appropriate course of management. History should include a review of all the pertinent risk factors, a description of initial symptoms, and an outline of the subsequent course. Examination should include evaluation of symptomatic areas and a careful neurologic examination.

Consultation is warranted in doubtful cases to help establish a diagnosis and in documented cases to help determine a course of management as outlined in Figure 1. While outlining general principles of care, we recognize that there is no substitute for the experienced judgment of the hyperbaric physician in the management of the individual patient with DCS and that liberal use of consultation is often helpful.

Prompt compression therapy is indicated for any patient who has symptoms or signs of DCS at the time of evaluation. We would exclude only those patients with minor cutaneous manifestations from this general rule. Observation at rest on 100% oxygen is <u>not</u> appropriate definitive care for DCS no matter how minor.

The patient with pain only bends whose symptoms clear on descent from altitude and who is asymptomatic at the time of evaluation does not require compression therapy. Observation for 2 to 4 hr is indicated. Supplemental oxygen is not necessary during this period, and the patient may be allowed quiet activities. If the patient remains symptom free, he can return to duty not involving flying. Exercise should be restricted for 24 hr.

Following an episode of DCS there is a period of increased susceptibility to DCS; this period does not exceed 48 hr. Consequently duty should not include flying for 48 hr following an episode of DCS. If no symptoms recur during that period, the patient can be safely returned to flying duty.

The patient with pain only bends who has symptoms at the time of evaluation requires prompt compression therapy. If symptoms clear before transportation arrives, transportation may be cancelled and the patient is observed for 24 hr. If no symptoms recur, he may be returned to flying duties in 48 hr.

One may be incautiously tempted to parallel this situation by voluntarily establishing a period of observation to see if symptoms clear at rest on 100% oxygen. Symptoms may clear or may get worse. In the event symptoms get worse, if subsequent treatment is prolonged or if residuals persist, the patient has been unnecessarily jeopardized; there is no support for the physician's decision to delay definitive treatment while he observes a symptomatic patient with DCS.

The patient with neurologic or pulmonary manifestations of DCS or vasomotor collapse should be transported to a hyperbaric facility for prompt compression therapy, even if symptoms resolve prior to transportation.

Recurrent pain only bends arouses increased concern since it indicates that bubbles are persistent and fibrin formation may be occurring around bubbles making subsequent resolution more difficult. Recurrent pain only bends warrants prompt compression therapy even if spontaneous resolution of symptoms again occurs before compression is initiated.

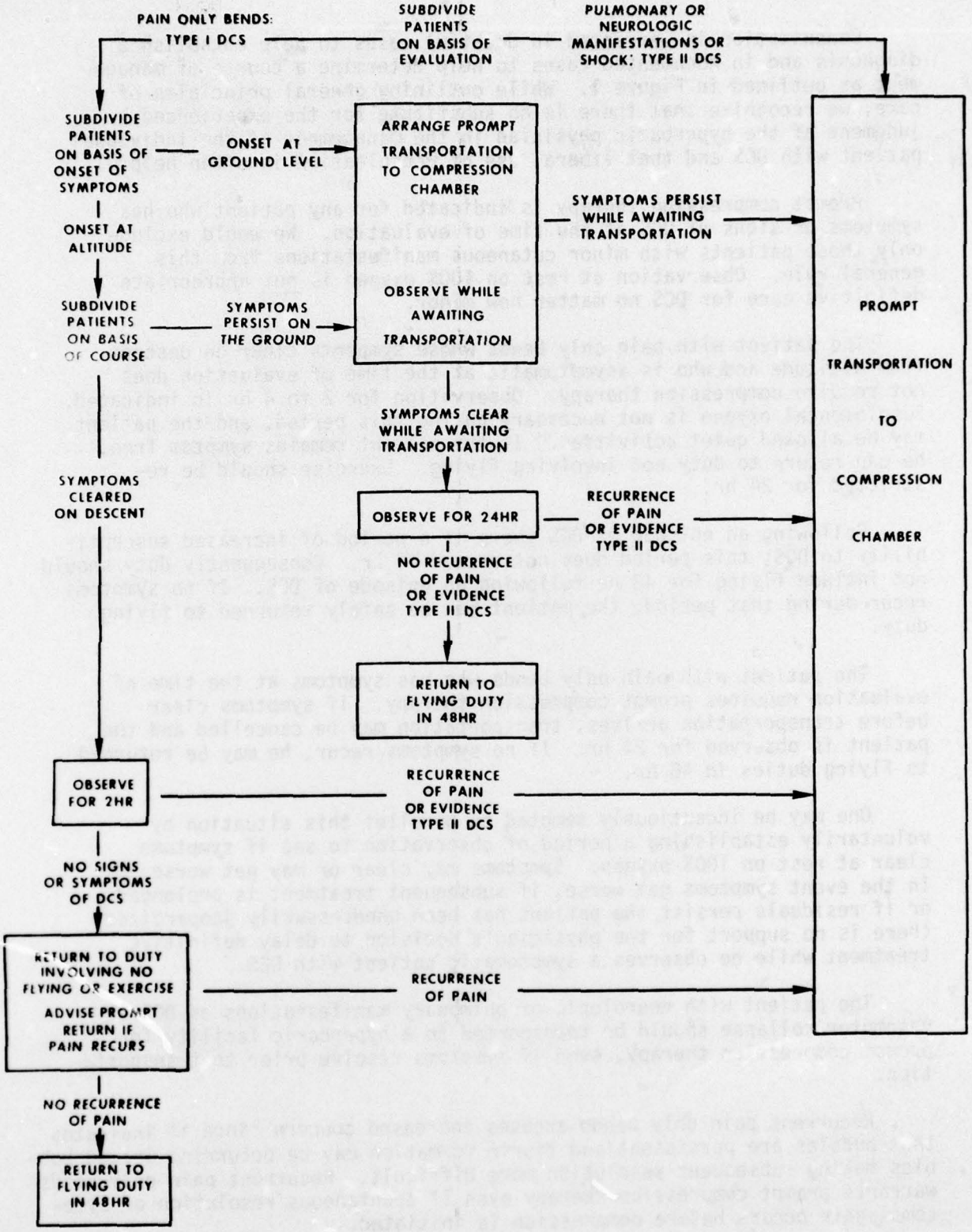

Figure 1. Decision points in the primary care of altitude decompression sickness.

Transportation to the Compression Chamber (Hyperbaric Facility)

When the decision to transfer the patient to a compression chamber has been made and coordinated with a hyperbaric facility, the patient should continue at complete rest, receive 100% oxygen by snug-fitting mask, and be kept under close observation while awaiting and during transportation. Aircraft used to transport patients with DCS should be pressurized to ground level. Any compromise which involves exposure to even slight decrease in barometric pressure may cause exacerbation.

ADJUNCTIVE THERAPY

An intravenous infusion to provide access to the circulation is warranted in patients with DCS because of the potential for vasomotor collapse. Lactate Ringers is a satisfactory solution for this purpose. In serious Type II DCS one may add a colloid solution such as 500 ml of low molecular weight dextran every 12 hr for 2 days to attempt to prevent or reduce rouleaux formation. One may elect to forego an intravenous infusion in a patient with minimal pain only bends as long as there is no evidence of progression.

Corticosteroids may be administered at the compression chamber to reduce cerebral edema. The associated increased risk of oxygen toxicity suggests that the use of corticosteroids in DCS should remain in the hands of the hyperbaric physician.

Since we recognize that the mechanical effect of bubbles may not be the sole factor in the pathophysiology of DCS, much attention has been given to adjunctive treatment that would address other factors. Anticoagulants have received prominent consideration in these investigations. Aspirin and heparin have been evaluated along with many other drugs. Opinions vary regarding the value of anticoagulants in the management of DCS (5, 22, 27, 32), and at present there is no clear consensus that anticoagulants have a role in the clinical management of DCS. Hopefully, continued investigation will resolve these issues and may establish a role for drug therapy in the definitive care of DCS.

PROGNOSIS

The immediate outlook for the patient with altitude DCS who receives prompt compression therapy is excellent. In a review of 145 cases of altitude DCS who were treated with compression therapy during the period from 1959 through 1976 there were no fatalities and only three patients had residual deficits (10). Of the 120 patients treated in a USAF hyperbaric facility only one had a residual deficit, a visual field defect.

Following an episode of DCS there is a period of increased susceptibility to DCS. This period does not exceed 48 hr and mild cases of pain only bends, which resolve prior to treatment and do not require compression therapy, can then be safely returned to flying. We know of no increased risk of subsequent DCS in these patients.

Serious cases of DCS present a more complex problem in the long-term prognosis for risk of subsequent DCS. Individual variability in susceptibility to DCS independent of known risk factors has been recognized. A severe case of DCS may represent an individual susceptibility to the disease. The possibility that an aviator with a serious case of DCS has an increased individual susceptibility raises real concern that he may have an increased risk for subsequent occurrence of serious DCS.

Concern over that risk forms the basis for the policy in the United States Air Force that DCS with neurocirculatory manifestations or DCS which required compression therapy is disqualifying for flying duties (2). Consideration of a waiver for DCS is based on a thorough evaluation of the circumstances that prompted the episode of altitude DCS and on the severity of manifestations and on the duration that manifestations persisted. Waiver is frequently granted to aviators with pain only bends who responded promptly to compression therapy.

Since serious cases of altitude DCS are often heralded by minor symptoms of pain only bends, an excellent prognosis can be achieved by expeditious primary care and prompt compression therapy when indicated.

SUMMARY

Evolution of bubbles in the tissues secondary to decreased barometric pressure plays a central role in the pathophysiology of DCS.

Exposure to altitude is the prime risk factor for bubble formation. Flying within 24 hr of diving carries an increased risk for altitude DCS. Additional risk factors include duration at altitude, exercise at altitude, age, and obesity.

Cabin pressurization and denitrogenation are the major factors in prevention.

Manifestations of DCS are often atypical. Consultation with a hyperbaric physician may be helpful in establishing a diagnosis and is essential in arranging for compression therapy.

Initial management of altitude DCS includes 100% oxygen, rest, and prompt descent. The only definitive treatment for altitude DCS is prompt compression therapy. Transportation should be pressurized to ground level. Prognosis is excellent in altitude DCS in the patient who receives prompt compression therapy.

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